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THE EFFECT OF VANADIUM ON THE INTENSITY OF OXYGEN CONSUMPTION BY GUINEA PIGS

[Vliyaniye vanadiya na intensivnost' potrebleniya kisloroda morskimi svinkami]

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T. Kh. Kuliyeva

The entry of a number of trace elements (iodine, manganese, zinc, cobalt, etc.) into biologically active products (enzymes, hormones, vitamins) alert one to approach the study of the physiological role with particular attention.

Vanadium is among the trace elements whose significance in this regard has not practically been studied. However, the constant presence of vanadium in plant and animal organisms indicates its possible participation in general biological processes. Its content in the environment and in organisms is extremely small: in soils — $1 \cdot 10^{-2}$%, sea water — $5 \cdot 10^{3}$%, plants — $1 \cdot 10^{4}$% animals — $1 \cdot 10^{4} + 10^{5}$% [4]. In certain animal organisms, its concentration occasionally reaches high values: the ascidians — from $4 \cdot 10^{3}$ to $5 \cdot 10^{4}$% [4], the sea cucumbers — from 2.5 to 1900 mg/kg [7].

As a catalyst, vanadium is widely used in the chemical industry and metallurgy in manufacturing alloy steel.

With respect to the animal organism, there are indications of an increase in the oxidation of phospholipids in a suspension of

rat liver [9] under the effect of sodium metavanadate. The authors have hypothesized that natural enzymes are present in the liver which contain vanadium as an active group. Subcutaneous injection of rats with sodium metavanadate ($NaVO_3$) in the amount of 1 ml 0.00125 mole causes an increase in their basal metabolism [5].

There are indications [8] of the capacity of vanadium pentoxide and ammonium vanadate in a dose of 1 mg/kg (in recalculation for vanadium) to reduce oxygen consumption in rats. In a dose of 0.5 mg/kg, vanadium pentoxide increases oxygen consumption; ammonium vanadate administered in the same calculation had no effect on the level of oxygen consumption. Disruption of the conditioned reflex activity in albino rats began to occur during the use of vanadium in doses of 0.05 mg/kg.

Presently, vanadium and fluorine are being given attention with respect to the appearance of dental caries [1]. An increased amount of vanadium is noted in rats which are most resistant in the pathogenesis of the caries lesion.

The physiological role of vanadium in the animal organism is still far from known. Its possible participation in the oxidative-reducing processes stimulated us primarily to investigate its effect on the intensity of oxygen consumption. In this case, it was necessary to limit its effect as a biotic factor (trace element) from its effect on the organisms as a toxicopharmacological agent. With this goal, we undertook systematic investigations of the effect of its various dosages on the intensity of oxygen consumption by guinea pigs.

The experiments were performed on guinea pigs (135 specimens) weighing from 250 to 400 grams. The experimental pigs (97 specimens) were divided into groups and were perorally administered spring water solutions of sodium metavanadate through a rubber tube in concentrations of 0.008, 0.025, 1, 40, 126, and 1600 mg% twice a day in the amount of 5 ml each; the control animals (38 specimens) received pure water.

The consumption of oxygen was measured in each guinea pig a day later in an enclosed type apparatus in the Miropol'skiy modification. The background consumption of oxygen was preliminarily established over 10 - 15 days; then, the guinea pig was daily administered one of the indicated solutions of sodium metavanadate. The results of these changes were corrected to normal pressure and a temperature of 0°. Oxygen consumption was calculated for one hour per 1 kg of animal weight.

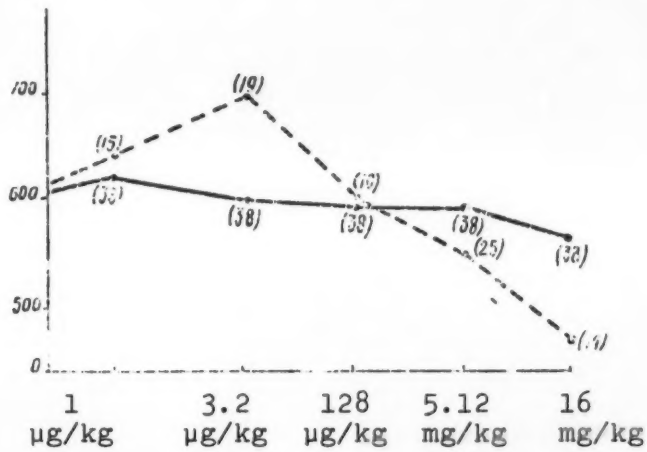

Figure 1. The effect of sodium metavanadate on the intensity of oxygen consumption by guinea pigs: on the ordinate — the amount of absorbed oxygen in ml per 1 kg of animal weight per 1 hour. On the abscissa — the amount of metallic vanadium per 1 kg of animal weight. Numbers in parentheses are the number of experimental animals. Data with dosages of 1, 3.2, and 128 µg/kg are given on the fifteenth day, 5.12 mg/kg — on the tenth day, 16 mg/kg on the fifth day after beginning vanadium dosage

The obtained data demonstrated the capacity of vandium to influence guinea pig oxygen consumption. This effect was dependent on dose. In a vanadium dosage in the calculation for the pure metal in the amount of 1 µg/kg animal weight per day (solution of 0.008 mg%, with dosage for 25 days), statistically reliable changes in oxygen consumption did not occur in comparison with the initial background ($n = 15$, $P < 0.1$).

In the group of guinea pigs which received a daily dose of vanadium of 3.2 µg/kg live weight (the 0.025 mg% solution), a statistically reliable increase in oxygen consumption was noted beginning with the 15^{th} day of dosage ($n = 19$, $P < 0.001$). But, with an increase of dosage to 128 µg/kg (1 mg% solution), statistically reliable changes in the oxygen consumption were absent over a 25-day period ($n = 10$, $P < 0.2$). However, in the group where sodium vanadate was given in a dose of 5.12 mg/kg (40 mg% solution), beginning with the 9^{th} day, a gradual, statistically reliable reduction in oxygen consumption was noted ($n = 24$, $P < 0.02$). A dose of 16 mg/kg (126 mg% solution) caused the death of 5 of 15 animals after the 5^{th} day of dosage. All of the surviving guinea pigs died on the 10^{th} day ($n = 25$, $P = 0.01$).

Before death, there was a sharp reduction in the degree of oxygen consumption in comparison with the values existing before the beginning of vanadium dosage. Diarrhea and paralysis of the posterior extremities were observed. At autopsy of the cadavers, plethora of all of the internal organs and capillarostases were detected. The liver and spleen were enlarged. Necrotic foci were found in the spleen with punctate hemorrhages in the gastric and intestinal mucosa.

A higher dose — 204 mg/kg live weight (1600 mg% solution) caused the death of the guinea pigs on the third day (experiment performed on 14 guinea pigs).

In the special group of control guinea pigs, the same intervals of time under the same conditions of observation did not produce a statistically reliable difference in the intensity of oxygen consumption between the first 10 days of observations, making up the initial background of oxygen consumption, and the subsequent 25 days, which comprised vanadium dosage in the experimental groups ($n = 38$, $P < 0.5$).

The cited data demonstrate the selective relationship of the guinea pig organism to small amounts of vanadium. It was reflected in the fact that certain extremely small dosages (a daily dose of 3.2 µg/kg) of vanadium caused the intensity of oxygen consumption to increase, beginning on about the 15th day of dosage. This effect was stable and continued throughout the course of the entire period of subsequent observations (15 - 20 days).

It is characteristic that the increase in the indicated dosage (128 µg/kg weight) did not cause an apparent effect. This dose was seemingly "ineffective". Dosages reduced to 1 µg/kg were also "ineffective". A significant increase in dosage (up to 5.12 mg/kg reduced the consumption of oxygen, and at a dosage of 16 mg/kg death of the animals was observed.

The noted characteristics of the reaction of the guinea pigs organism to different doses of vanadium show the existing "zonality of action" [2]. A small dose of vanadium (3.2 µg/kg), to one degree or another approximating its natural content in the environment and exerting a stimulating effect on oxygen consumption, is the basis to view it as biotic, i.e., the dosage in which this trace element emerges as a factor characteristic of the organism which influences its internal systems by participating in the biochemical processes.

Increasing the vanadium dosage to 128 µg/kg had no effect, obviously because of activation of the protective function of various physiological barriers that prevent the excessive entry of this trace element into the organism (the "zone of effectiveness"). A significant (up to 15.1 - 16.0 mg/kg) increase in dosages, which have a clearly toxic effect, acted on the organism according to the principle of overcoming the resistance of its protective barriers (the "zone of toxicopharmacological effects").

The concept given above regarding the zonality of effect of trace elements has been specifically proven in a special investigation devoted to studying the effect of different doses of copper

on the formation of nucleic acids in the liver [11], and the selective relationship to small amounts of chromium (0.1 - 1.0 µg per 1 ml) in the study of Mertts-Roginskiy [10].

The phenomenon of "zonality" of the effect of trace elements may present not only theoretical, but also practical, interest in solving the problem of the selection of doses of a trace elements, specifically vanadium, as a stimulator of physiological processes.

The toxic doses of vanadium for guinea pigs which we noted indicate the relatively high sensitivity of the guinea pig organism to this trace element.

Conclusions

During daily (for 15 - 25 days) double administration of an aqueous solution of sodium metavanadate perorally in guinea pigs, the following were noted (with respect to the intensity of oxygen consumption): the absence of an effect with a daily dose (in calculation for the metal) of 1 µg/kg; an increase in oxygen consumption at a dose of 3.2 µg/kg ("zone of biotic effect"); the absence of a statistically reliable effect at a dose of 128 µg/kg ("zone of ineffectiveness"); a toxic effect at a dose of 5.1 mg/kg or higher ("zone of toxicopharmacological effect").

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